

## ANALYSIS OF SURVEY ON DIABETES IN CHILDREN

By FRANCIS SCOTT SMYTH \*

*The prognosis of juvenile diabetes before the introduction of insulin was invariably fatal. Since the advent of insulin the outlook is far more hopeful, and certain patients have shown some recovery of tolerance. The treatment should include the optimum caloric and protein intake and a safe fat-carbohydrate ratio. Vitamins should be emphasized, cod liver oil, green vegetables, and heliotherapy are advocated. Insulin should be given in sufficient amount to keep the blood sugar approximately normal even to the risk of occasional hypoglycemia. All foci of infection should be removed. All children with doubtful symptoms should have a glucose tolerance test, elimination of foci of infection, and should be kept under observation for an indefinite period as is done with congenital luetics.*

Discussion by Horace Gray, Santa Barbara; William W. Belford, San Diego.

LITERATURE about juvenile diabetes is scant. This is partly the result of inaccurate methods of diagnosis and partly the disinterest due to the fatal prognosis invariably found before insulin was available. Such patients as were seen or reported before the use of insulin usually showed a pitiful picture of malnutrition and dwarfism.

This study of diabetic patients from the University of California and Children's hospitals is a review of the possible factors responsible for diabetes, contrasting the prognosis under modern treatment with that found in the preinsulin period. Thirty-one patients are listed under the diagnosis of diabetes, of which nineteen were true diabetics. In the remaining twelve the diagnoses are questionable and they are included as border-line cases, three of whom had symptoms of diabetes insipidus. Inasmuch as the symptomatology of all the border-line cases was very similar to that of the true diabetic onset, and since the early diagnosis of diabetes is most essential, the border-line cases represent a very important class for preventive work and follow-up.

*Review of Cases*—Of the 19 unquestionable diabetics 5 were females and 14 males, which very closely approximates the generally recognized sex proportion of 3.1. The oldest patient was 13 years, the youngest 2 years, four being under 4 years of age. Five gave a positive family history of diabetes. The remaining fourteen had no such history, though of course, the accuracy with which a history was obtained was variable and in no instance was an adequate history of events subsequent to diagnosis obtained. Of the 5 with positive family histories only 1, a sister, was of the patient's generation; 3 of the 5 were in males and 2 in females. These familial histories are of interest in view of the proportion Hansen has reported in his recent article

on the rôle of heredity in diabetes and which is in contrast to the findings of Knox, who claims in his report on diabetes in infancy that heredity plays but little part.

In addition to the family history the following possible predisposing causes were obtained from the patients' records: two cases of congenital developmental failure, one had obvious glandular dystrophy and the other cleft palate, which is simply taken as a possible indication of other maldevelopments. Three patients showed previous dietary disorders, two of excessive carbohydrate and one of prolonged difficult feeding. The rôle of acute infections, discussed in detail below, is indicated by the following figures: upper respiratory infections (tonsils) 7, influenzal attacks 2, exanthemata 23, measles 9, parotitis 4, varicella 4, pertussis 4, scarlet fever 1, diphtheria 1, gastrointestinal upset 4, frequently accompanied by severe abdominal pain of possible significance in involvement of the pancreas.

Primary symptoms varied with the adequacy of parental observations. For example, it would be supposed that polydipsia would precede emaciation as the first symptom. However, 4 showed polydipsia as the first symptom, 5 fatigue, 3 polyuria, enuresis or nocturia, 3 loss of weight or emaciation, 1 complained of itching skin and in 1 glycosuria was accidentally discovered unpreceded by observed symptoms, in 2 no record was made of primary symptoms. The fully developed symptomatology included polyphagia, polydipsia, polyuria, and loss of weight in all but three patients, in whom polyphagia was not found; these were all under 7 years of age, two males and three females.

*Focal Infections*—A striking finding was the presence in sixteen of chronic foci of infection, tonsils 12, teeth 4, sinuses 2, middle ear (otitis media) 2, skin 2, mastoid 1, vaginitis (nonspecific) 1, not reported 3. Attempts at removal of the foci of infections were made in five. It is reasonable to assume that infection materially handicaps the recovery of autogenous tolerance and that the prognosis is infinitely improved by removing the foci.

The *treatment* of the nineteen patients was so varied that except for the advent of insulin a summary would be too detailed. Follow-up records show that 8 are dead, 10 alive, and 1 patient was not located. Of the patients who died five had never received insulin. The one not found was in the preinsulin period and is assumed to have shared in the 100 per cent mortality of the five patients treated before insulin was available. Of the three deaths which occurred subsequent to the use of insulin one died in coma after 10 units of insulin had been given for a 10-year-old child with a .5 blood sugar. In another patient the parents refused to allow insulin after discharge from the hospital and the child died in coma at home. The third patient was allowed frequent dietary indiscretions by the parents and also received insulin irregularly; death in coma occurred at home.

The highest blood sugar recorded on entry was .760, lowest .228; highest  $\text{CO}_2$  on entry 36.8, lowest 15.2.

*Border-Line Cases*—Of the twelve border-line cases there were nine males and three females, which

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again agrees with the generally accepted findings. The oldest patient in this group was 14 years and the youngest 14 months. There was a family history of diabetes in three females and three males; one in the fourth generation, four in the third generation, and one in the preceding generation. Of predisposing causes other than heredity there were four with congenital developmental failure of whom three had obvious glandular dystrophy and one had mental deficiency. Acute infections were as follows: upper respiratory 4, influenza 1, pneumonia 1, gastrointestinal upsets 2, measles 4, scarlet fever 1, pertussis 3, and diphtheria 1.

**Primary symptoms:** Eight had polyuria, 2 polydipsia, 1 fatigue, and in 1 glycosuria was accidentally discovered. **Entire symptoms:** 1 developed polyphagia, polyuria and polydipsia, and 3 had glycosuria; loss of weight was fairly common.

**Focal infections** included chronic diseased tonsils 6, dental caries 3, sinus infection 1, and not recorded 2.

**The Follow-Up**—Two of the twelve under observation became definitely diabetic, one had a diabetic syndrome associated with glandular dystrophy, two had a definite glandular dystrophy unassociated with complete diabetic symptoms, two are still under observation, two have made apparent recovery, and three have not been heard from.

**Laboratory Findings**—Blood sugars on entry were all within normal limits. A glucose tolerance test was done in eight, four were diabetic in character and two of these later became the true diabetics, two are still under observation but the glucose tolerance has as yet not been repeated, three were atypical tests but not diabetic in character (two of these became definitely glandular troubles, one a glandular dystrophy with diabetic symptoms), and one was normal. In one of the patients (L. H.) in whom diabetes was later diagnosed a tonsillectomy was done, and it is interesting to note the improvement in the glucose tolerance test subsequent to tonsillectomy, but before the final diagnosis of diabetes was made.

**Discussion**—The factors responsible for diabetes are not definite, but certain influences probably contribute to its production. The well-known racial predisposition to the disease in Hebrews decreases in American and European Caucasians, and is seen less frequently in the colored races. Certain definite familial tendencies are seen, but this does not follow the Mendelian laws of inheritance in any clear-cut manner (Hansen, *Ungesk. f. Laeger.* 86:341-44, 1924). Faulty diet has been frequently suggested as being etiologically important in diabetes. Modern civilization, with its ever increasing consumption of concentrated carbohydrate foodstuffs, might be held accountable for the increasing incidence of the disease. A long-continued overbalanced carbohydrate diet may materially affect the tolerance of carbohydrates, and two of our diabetics gave such a history. Many diabetic patients show a definite relation to acute infections. This is well illustrated in the cases reported by Stengel (*Contributions to Med. and Biol. Research*, dedicated to Sir William Osler, 2:1186, 1919) in which the diabetic syndrome appeared and disappeared with recurring in-

fections. Some patients give a history of infection preceding the symptoms of diabetes, though as a rule the damage from the infection is so severe as not to be relieved when the infection disappears. Reasoning from the reaction of diabetics to infections incurred subsequent to the onset of the disease we are at once confronted with the strikingly deleterious influence of infection on pancreatic function. The peculiar finding of Holsti (*Ztschr. f. Klin. Med.*, 20:272, 1892) in which influenza preceded the onset of diabetes in many of the patients studied, and the findings of Schloss (personal communication) of disturbed carbohydrate metabolism in influenzal infection might indicate some specificity with regard to its influence on the pancreas. Profound ketosis is encountered with seemingly light influenzal infections of the upper respiratory tract. Of the acute exanthemata, mumps has perhaps been most often found in association with pancreatic upset. Garrod (*Lancet*, 1:557, 1912) has reported twenty-two cases of mumps with diabetic symptoms, and Patrick (*British M. J.*, 2:802, 1924) has also reported acute diabetes following mumps. However, diabetics are seen in whom none of the above mentioned influences operate.

We are more or less forced to assume that there may be individuals in whom diabetes represents a failure of development of pancreatic tissue somewhat analogous to thyroid subdevelopment. Pathological studies substantiate this in some cases, but in many we are unable to correlate the pathological picture with the clinical dysfunction. Osler (*Modern Medicine*) reported persistent glycosuria in a newborn. The well-known association of diabetes with internal gland disorders is again indicative of congenital maldevelopment. Inasmuch as we frequently find congenital anomalies together, we are not surprised at the findings of at least one such congenital defect in our series. Poynton and other English writers have also noted the occurrence of diabetes with celiac disease, which also probably represents a congenital developmental failure. In the presence of such a congenital pancreatic failure a seemingly slight infection might bring the latent diabetic symptoms to their threshold.

In infants and young children diabetes is often ushered in with an acute gastrointestinal upset. Vomiting and diarrhea may be characteristic, and the abdominal cramp-like pains may be of significance with regard to pancreatic involvement. With the subsidence of the gastrointestinal symptoms, increased thirst and polyuria may persist, and if the significance is not quickly recognized, may lead to a rapid and fatal termination. Unlike adults a failing appetite may be associated with diabetes in younger children, and parents are prone to ascribe emaciation, which is a common finding in this disease, to the failing appetite rather than its true cause. The skin becomes very dry and occasionally xanthoma may be found. In children polyuria is perhaps the first symptom noticed by the parent, who may have been attracted to the condition by bed wetting or enuresis. In one case which came under my observation the parent first noticed the crystalline appearance of the dried urine around the toilet. In general, however, after 7 years of age

the diabetic child's full symptoms are similar to those of the adult.

#### DIAGNOSIS

In the presence of the classical symptoms, polyuria, polyphagia and polydipsia with marked loss of weight, the persistent reduction of Benedict's solution by the urine is sufficient to assure the diagnosis. However, since the classical symptoms may be lacking in infants and young children, and since the prognosis depends so largely on an early recognition of the disease, an effort should be made to rule it out whenever there is the slightest suspicion of diabetes. This is possible through the newer methods of laboratory diagnosis. Glycosuria itself is not sufficient evidence for a diagnosis, as this may appear in a variety of other conditions, for example, alimentary glycosuria, pentosuria, renalglycosuria, concentrated urine, anhydremia, chronic nephritis, and cachexia. A single blood-sugar estimation is likewise insufficient evidence for a diagnosis, though perhaps it is of more value than ordinary urinary tests since there may be a high renal threshold. The accurate diagnosis of diabetes rests on the glucose tolerance test by which these other disturbances may be ruled out. Blood-sugar estimations are of value in the control of insulin therapy. With the development of diabetic acidosis, further analyses are of value. The carbon dioxide content of the plasma gives an index of the degree of acidosis far more accurately than the qualitative test for ketonuria.

#### TREATMENT

Before the advent of insulin the outlook for infants and children with diabetes was almost hopeless. Through properly administered insulin and a regulated diet the prognosis has been greatly improved. Certain aspects of the treatment differ from that in the adult. In children we have growing organisms with a greater caloric requirement and a growth requirement which must be met with sufficient food. They may be capricious in their appetites and devoid of an appreciation of the nature of their ailments. A treatment which does not permit as normal a development as possible is not wholly successful. Since the introduction of insulin the prolonged starvation treatment has not been necessary and more nearly normal development is therefore possible. A diet must be obtained which is sufficient as to calories and nitrogen content, palatable and safely balanced in antiketogenic ratio, and it must be remembered that this latter is subject to individual variations (Levine and Wilson, *Am. J. Dis. Child.*, 31:323, 1926). With such a diet sufficient insulin must be used to keep the blood sugar within normal limits.

The initial insulin dosage is reckoned from the blood-sugar content. The body fluid will approximate two-thirds of the body weight. The excess sugar over the normal content can then be ascertained and insulin given to metabolize this. As a rule the excess blood sugar if metabolized by sufficient insulin will take care of the acidosis present. In extreme acidosis, however, additional therapy may be required. Where there is marked dehydration and anuria a hypodermoclysis of saline should

be followed by the intravenous administration of a glucose solution. Joslin has criticized the use of intravenous glucose, but its function in this instance is not so much for its antiketogenic action as for the more rapid absorption of the necessary fluid and a very prompt stimulus to diuresis. Another disputed therapeutic measure is the use of sodium bicarbonate. In acidosis when the  $\text{CO}_2$  content of the blood is reduced the administration of sodium bicarbonate will raise it. In the average patient with only a moderately reduced  $\text{CO}_2$ , insulin alone is sufficient to metabolize excess ketone acids and raise the  $\text{CO}_2$ , in which case administration of sodium bicarbonate would merely confuse the picture. Where, however, the  $\text{CO}_2$  is less than 15 volumes per cent, there may be an actual depletion of the base in the blood, and sodium bicarbonate administration would then be indicated.

The diet is based on the ideal weight, the caloric requirements and nitrogen need for weight and age. From the nitrogen (protein intake) the minimum carbohydrate necessary to prevent ketosis may be reckoned from Shaffer's formula, although we have found that patients may show an individual variation in the ketogenic ratio. Palatability often demands a slightly greater amount of carbohydrate than the calculated minimum, since the diet must be made agreeable for the patient. The fat content of the diet is fairly high but safe, and gives very satisfactory results. The vitamin content of the diet is emphasized. Cod liver oil and greens are essentials. In infants special formulas with reduced sugar, curds, etc., must be used. Continued treatment and favorable prognosis demand strict adherence to the diet and analysis of each individual specimen of urine. While in the hospital blood sugars are obtained from time to time by which the renal threshold is approximately estimated, and though this is subject to variation it is of value in the further treatment of the patient.

In infants insulin shock must be carefully guarded against. Drowsiness or marked perspiration are perhaps the most common early findings. If orange juice is not promptly administered they may develop convulsions or coma. In older children the subjective symptoms may be indicative of the imminence of a hypoglycemia, but one should be on the lookout for malingering. Nevertheless, if possible, the blood sugar should be kept low, even running the risk of hypoglycemia if the maximum regeneration of the pancreas is to be expected. Ulrich (*J. A. M. A.*, 83:1914-1915, 1924) even recommends hypoglycemia as allowing the maximum regeneration. Any foci of infection such as the teeth, tonsils, and sinuses should be properly treated, and invariably the response in tolerance justifies these measures. The occurrence of a high per cent of upper respiratory infections has been shown in this study, and those patients who had the foci removed showed a distinct increase in carbohydrate tolerance. While in the hospital, Alpine lamp treatments are advisable, and sun baths are advocated on discharge.

#### PROGNOSIS

The prognosis of diabetes in children treated with insulin is yet to be written. So far the results have

been very encouraging. The previous starvation and low calorie diet treatments were invariably accompanied by malnutrition and poor development. Allen still adheres to his "undernutrition" treatment since the advent of insulin. I feel that although the Allen treatment was an improvement over methods previously in vogue, undernutrition *per se* is not an asset but a hazard. Overweight is not advocated, but the child should be approximately at its ideal weight. With adequate diet and insulin they approximate normal development. Infections and major operations may be tolerated without extreme danger.

With regard to their recovery of carbohydrate tolerance certain features are important. The earlier the treatment is started the better the chances of recovery of tolerance. Acutely ill patients promptly treated usually show a more rapid recovery than those of longer duration. The elimination of foci of infection materially improves tolerance. Finally, a strict adherence to diet, sufficient insulin to keep the urine sugar-free and the blood sugar normal undoubtedly offers a better chance for the organism to repair the damaged tissue. There is considerable indication from experimental studies (Copp and Barclay, J. Metabolic Research, 4:445-51, 1924) to justify the assumption that insulin may act somewhat like a splint to a fractured limb, affording a chance for regrowth of insulin forming tissue if properly applied.

#### SUMMARY

The prognosis of juvenile diabetes before the introduction of insulin was invariably fatal. Since the advent of insulin the outlook is far more hopeful, and certain patients have shown some recovery of tolerance. The treatment should include the optimum caloric and protein intake and a safe fat-carbohydrate ratio. Vitamins should be emphasized, cod liver oil, green vegetables and heliotherapy are advocated. Insulin should be given in sufficient amounts to keep the blood sugar approximately normal even to the risk of occasional hypoglycemia. All foci of infection should be removed. All children with doubtful symptoms should have a glucose tolerance test, elimination of foci of infection, and should be kept under observation for an indefinite period as is done with congenital luetics.

I wish to thank members of the staff of the Children's and University of California hospitals for courtesies and help to make this study useful.

#### DISCUSSION

HORACE GRAY, M. D. (Santa Barbara Clinic, Santa Barbara, California)—Passing over the three patients with diabetes insipidus, which is hardly related to diabetes mellitus, there remain nineteen cases diagnosed definitely diabetes mellitus and nine called *questionable*. This last group is interesting both because it forms so large a fraction of the whole series reported and because doubtful and cured cases in children have curiously enough received very little attention in the literature. Probably all nine cases, certainly the two border-line cases "having made apparent recovery," would be worth tabulation in respect to their symptoms at the worst stage observed, for example, the highest degree of glycosuria and of hyperglycemia found in each case.

The cases with onset following *gastrointestinal* attacks, especially the four with abdominal pain, again form a group of special importance, inadequately covered in the literature. Tabulation of these would be interesting, espe-

cially as regards the interval between the gastrointestinal upset and the diagnosis of diabetes.

The *blood sugar* maximum 0.760 and minimum 0.228 per cent seem high in view of the frequent observation in children of low blood sugar with even severe glycosuria. A table of the blood sugars found in the twenty-eight patients would therefore be worth having on record.

Regarding the distribution of *diet* in children, Doctor Smyth appears to favor the minimum carbohydrate, in other words, a high fat diet. It might be of interest to report the average diet given to his children expressed in grams of carbohydrate, protein, and fat per kilo of body weight, thus permitting comparison with diets fed in other clinics.

The rôle of *acute infections* as stated in the paper gives the impression that there were fifty-nine cases in the series or else that several patients had several infections in the past. The latter seems to the reviewer of no significance because, if the infections named merely occurred at some time in the past history of the children, their connection with the diabetes seems too vague to be worth discussing. One would prefer to know the name of the infectious disease, if any, which occurred within three (or perhaps six) months before the onset of the diabetes.

The *sex frequency* noted is striking in view of the statement on page 125 of Joslin's book that only 57 per cent of the cases were males, both in his series and in that of Schmitz, which are by far the largest series reported; and that among the diabetic deaths in the United States only 45 per cent were males.

WILLIAM W. BELFORD, M. D. (Electric Building, San Diego)—The absence of the characteristic symptoms of diabetes in so many cases, both the proven and the border-line, and especially in the primary symptoms of those patients under 7 years, is striking. Careful routine urine examination is to be emphasized.

Doctor Smyth's outline of treatment works admirably. The calculation of the diet is simple and the amount of protein, fat, and carbohydrate is such that the diet is quite palatable. That tolerance of some degree is recovered is hardly to be doubted when one sees smaller and smaller doses of insulin given and the patient showing no glycosuria. Those with no tolerance or "total" diabetes are difficult to keep sugar-free and often have insulin reactions. Lapses in the care of the prescribed diet or the occurrence of an infection rapidly lowers the recovered tolerance which is not regained, all too often, when the diet is corrected or the infection overcome.

That the simplest foci of infection are not to be ignored in the diabetes of childhood cannot be too strongly stressed. Carious teeth, infected tonsils or sinuses must have attention before the tolerance for carbohydrates is completely lost. As to prognosis, it is by no means bad when the parents are thoroughly co-operative. When a boy of four weathers an acute appendicitis with peritonitis, one is well pleased. It is truly remarkable when it is learned that this same sturdy boy has been a total diabetic since the onset of the disease before 2 years of age and, in addition, has had bronchopneumonia and otitis media. Diet and insulin regulations were religiously followed by the mother, and the boy suffered no particular hardship.

DOCTOR SMYTH (closing)—The diabetes insipidus cases were included in the group because on admission they were diagnosed diabetes mellitus and as such presented certain symptoms similar to diabetes mellitus.

The above series of cases is so small as not to be comparable to the larger summaries of Joslin and others. A table of blood sugars for the entire group would be extremely valuable, but as some of the material was taken from records of several years past, accurate data were frequently wanting. Acute infections, especially if occurring in succession, appear to us of more than vague significance, although the time relation to the onset of diabetes sometimes should be mentioned.

As to diet distribution in children, it has been our plan to use fairly low carbohydrate. It was mentioned that the individual ketogenic ratio is the guide to the fat adjustment. We believe the possibility of reducing the insulin to a single injection or even eliminating it altogether is greater with low carbohydrate diets. This materially

aids in the prognosis where subsequent care is entrusted to the family. Where, however, the requirement for carbohydrate and the severity of the case require large doses of insulin, the patient resembling a chronic adult diabetic, there would be no contraindication for the high carbohydrate values as advocated by the Santa Barbara clinic. In children, however, the chances of recovery of autogenous tolerance seems to warrant a lower carbohydrate intake with corresponding lower insulin requirement.

## LIVING TRICHINELLA THIRTEEN YEARS OLD

By NEWTON MILLER \*

(From the School of Medicine, University of Utah)

DISCUSSION by Marshall C. Cheney, San Francisco; Alfred C. Reed, San Francisco; John V. Barrow, Los Angeles; Robert T. Jellison, Salt Lake City, Utah.

THE biology of this species is interesting and complicated. The adult males measure 1 to 1.5 mm. and the females 3 to 4 mm. in length. They are slender and taper from posterior to anterior end, which is pointed. The adults live for a few weeks at most in the intestines of carnivorous mammals, usually the rat, mouse, hog, or man. Here they mate; the males soon die and the females, after burrowing into the intestinal mucosa, give birth to many young, estimated at 1500 or more each. The larvae, thus discharged into the mucosa and submucous lymph, eventually reach the blood by way of the thoracic duct and are carried to all parts of the body. They leave the capillaries in the voluntary muscles and wander out among the muscle fibers. Others perchance reach the muscle by way of the lymph vessels or directly by burrowing through the tissues. After reaching the muscles, especially the diaphragm, intercostals and the muscles of the neck, tongue and eyes, they penetrate the sarcolemma, coil up and become inactive. Some are said to become imbedded among the muscle fibers. The invaded muscle fibers degenerate, connective tissue capsules form about the larvae and these capsules later become calcified. Thus imprisoned they may remain alive but inactive for months or years until the death of the host after which they may be eaten with the flesh of the host. The mammal ingesting the infected flesh of the first host becomes a second host for the encapsulated worms. The calcareous capsules are dissolved by the gastric juice in three to four hours and the worms set free to be carried into the intestine. Here they grow, reach sexual maturity in three to four days, mate and produce a brood of larvae which will become imbedded in the muscles of this host. The adult males die in a few days after being liberated in the stomach, and the gastrointestinal tract is free of both larvae and adults in about six weeks. Thus the cycle has been and may still be repeated *ad infinitum*. Rats are common carriers and, since they frequently eat their own dead, the trichina life-cycle can be repeated indefinitely within the bodies of these rodents. Among the animals devouring rats are the hogs. We learn from

United States reports on meat that 2 to 3 per cent of all pork marketed for exportation is infected with the trichinae. Fifteen per cent of all hogs raised in some localities within the eastern states are said to be parasitized. The human being eats pork and thereby hangs a tale.

Thirteen years ago Mrs. E. L., then a girl of 18, and her sister living in Laramie, Wyoming, ate some improperly prepared pork sausages. In fact they had eaten of the sausage on their way home from the meat market. The girls experienced no immediate ill effects, but on the fourth day both developed severe abdominal pains and cramps accompanied with severe diarrhea. The elder sister recovered from these symptoms in eight or ten days and has suffered no subsequent ill effects. The younger sister, Mrs. E. L., had scarcely recovered from this attack when she experienced further distress, as stiffness and intense generalized pain in the muscles very similar to acute muscular rheumatism, accompanied by marked anemia, puffiness of the face, edema of limbs, and fever. The condition became severe and the life of the girl seemed jeopardized. However, during the fourth or fifth week after eating the sausage the muscular pain and tender-

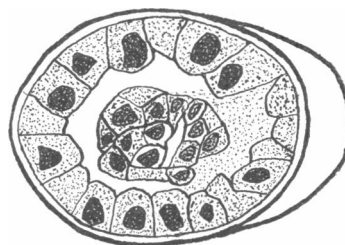


Fig. 1.—Camera lucida drawing of a cross section of a *T. spiralis* removed from Mrs. E. L. X 1500.

ness began to subside and eventually disappeared completely.

The girl grew to womanhood, married and raised a small family. She has not suffered any further pain or discomfort incident to the infection of thirteen years ago. The lady, now Mrs. E. L., entered the Salt Lake County General Hospital November 9, 1923, for the purpose of undergoing an abdominal operation. In taking the routine history the above-mentioned data were obtained which are given as the patient recalled the events. The operation was performed November 15, 1923. An examination of the incised muscle (right rectus abdominis) revealed to the naked eye the capsules of the encysted worms in abundance. In fact the meat inspector's expression "measley pork" could well be paraphrased "measley homo" and applied to this patient.

A piece of the right rectus was excised and more carefully examined. An estimate of fifteen to twenty worms per cubic centimeter of muscle was made. Pieces of the muscle, after being treated with dilute hydrochloric acid, were teased and examined microscopically. The outlines of the entire worms were nicely revealed, and it was plainly evident that the worms had not been absorbed nor were they calcified.

The next question then arose, were the worms still alive? To answer this question other pieces of the muscle were prepared by the usual histological tech-

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